STOP HEART ATTACKS NOW!

Lysine

and

The Pauling Therapy

Advanced Heart Technology

Vitamin C, Lysine and "Lysine Analogs" are Supported by U. S. Patent to Reverse Atherosclerotic Plaque Build-ups in Organs and Arteries caused by Lipoprotein-(a). (U. S. Patents # 5,230,996 and # 5,278,189)

- Read about a famous Nobel Prize-winning scientist's little known invention to fight heart disease.
- Discover a non-invasive heart disease therapy that is based upon essential vitamins and amino acids; i.e., "body safe" natural substances that are required for life in smaller amounts and do not require a prescription from your doctor.
- Learn why vitamins A, C, and E anti-oxidants and important amino acids are required for collagen production and healthy cardiovascular function.
- Realize that there are now more than 1100 mainstream scientific and medical journal articles that document the risk of heart disease caused by Lipoprotein(a) cholesterol.

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"Now I've got to the point where I think we can get almost complete control of cardiovascular disease, heart attacks and strokes by the proper use of vitamin C and lysine. It can prevent cardiovascular disease and even cure it."
Linus Pauling, British Journal of Optimum Nutrition (JON), Aug. 1994

CONGRATULATIONS! You are about to become more informed and take charge of your health and well being. This booklet contains controversial information. You will learn about **lipoprotein-(a)** (small -- "a") cholesterol, or **Lp(a)** for short. While you may not have heard of **Lp(a)** yet, **vitamin C** and the amino acids **lysine** and **proline** are the building blocks of collagen; the protein that literally holds your body together. All these substances promote healing. They may have other totally unexpected benefits as well. The only scientist to win two unshared Nobel prizes said:

> *"You need lysine to be alive, it is essential, but you can take lysine, pure lysine, a perfectly non toxic substance in food [as supplements], and that puts extra lysine molecules in the blood. They enter into competition with the lysyl residues on the walls of arteries and accordingly count to prevent lipoprotein-(a) from being deposited or even will work to pull it loose and destroy atherosclerotic plaques."* [Linus Pauling, JON, Aug. 1994]

According to this world famous chemist and medical researcher, sufficient amounts of **vitamin C** and **lysine**, dubbed the *Pauling Therapy*, may prevent and may even reverse atherosclerotic plaque build-up caused by lipoprotein-(a).

The NEW, Noninvasive Heart Technologies are:

- ***DRUG FREE!*** You do not need a prescription from your doctor to take advantage of this invention.
- ***LOW COST!*** Compared to prescription alternatives.
- ***HEALTHFUL!*** These natural substances are commonly found in the diet and are necessary in some amount for life itself. They promote general health by helping to heal and to strengthen your blood vessels and to increase muscle strength and fitness.
- ***ADVANCED SCIENCE!*** The new technology *increases* the dosage of these substances to high levels. This idea is based on the invention and the many careful experiments conducted by a team headed by one of America's most highly regarded scientists. The **Lp(a)** threat is now documented in over 1100 research papers.

Linus Pauling and Dr. Matthias Rath, MD, were awarded <u>two</u> United States patents for methods that reverse occlusive cardiovascular disease by inhibiting the binding of lipoprotein(a).

IS HEART DISEASE A VITAMIN DEFICIENCY DISEASE?

According to the Linus Pauling *Unified Theory of Heart Disease* the primary cause of cardiovascular disease (CVD), also called atherosclerosis and leading to heart attack and stroke, *is a chronic deficiency of Vitamin C in our diets.*

The Pauling/Rath hypothesis rests on the pioneering work of two Canadian medical doctors J. C. Paterson, MD and G. C. Willis, MD conducted in the late 1930s through the 1950s. These two medical doctors suspected that the heart disease process is related to the stability of arteries. They noticed that atherosclerotic plaques are not randomly distributed. Plaques usually develop at the same locations in places where blood vessels are stretched or squeezed, i.e., where mechanical forces and stresses are great, e.g., in the coronary arteries.

In 1937, on a hunch, Dr. Paterson measured tissue levels of vitamin C in hospital patients. *He showed that heart patients have 80% lower tissue levels of vitamin C in their blood vessels compared to other patients in the hospital.* In the 1950s, Willis showed that vitamin C intake correlates to atherosclerosis in both humans and guinea pigs. Willis discovered that 500 mg of **vitamin C** added to the diet could reverse plaque build-up in about half the human patients. *Plaque was not reduced*

in the controls. Daily **vitamin C** was 100% effective preventing atherosclerosis in guinea pigs.

Incomprehensibly these important first steps reported in the Journal of the Canadian Medical Association were not followed-up by other medical researchers.

The hypothesis that heart disease results from a chronic **vitamin C** deficiency flies in the face of drug company research programs.

Today reportedly more than 80 drug companies are seeking FDA approval for heart medications. Many are aimed at reducing the so-called "bad" LDL cholesterol.

LINUS PAULING VIDEO ON HEART DISEASE: UNIFIED THEORY OF CAUSE AND CURE.

In 1993, at 92 years of age, two-time Nobel Laureate Linus Pauling delivered a major one-hour speech that presents his *Unified Theory of Cardiovascular Disease.* In Pauling's opinion, heart disease can now be "completely controlled" by large amounts of **vitamin C** and **lysine.** The information on this video is yet to be publicly challenged, refuted, or even seriously questioned by any competent scientist or medical researcher.

Vitamin C, also called ascorbic acid, is essential and required for life. According to Elson M. Hass, MD:

> "*One important function of vitamin C is in the formation and maintenance of collagen, the basis of connective tissue, which is found in skin, ligaments, cartilage, vertebral discs, joint linings, capillary walls, and the bones and teeth. Collagen, and thus vitamin C, is needed to give support and shape to the body, to help wounds heal, and to maintain healthy blood vessels. Specifically, ascorbic acid works as a coenzyme to convert proline and lysine to hydroxy-proline and hydroxy-lysine, both important to the collagen structure.*
>
> http://www.healthy.net/library/books/haas/vitamins/cvit.htm

The classic vitamin C deficiency disease, **scurvy**, results from an *acute* deficiency. A human being on a daily diet of less than 10 mg of **vitamin C** will die from scurvy in weeks. One half of all scurvy deaths are the result of internal bleeding as the weakened blood vessels rupture. *The scorbutic guinea pig also suffers atherosclerosis.*

Pauling's crucial insight is that our diets lead to heart disease, *but not in the way most people think.* Our diets have sufficient **vitamin C** to prevent scurvy, but *less* than what we need to function optimally and keep our blood vessels flexible and strong. The result is a sub-clinical form of scurvy, i.e., *chronic scurvy.*

Linus Pauling believed that most of us suffer **chronic scurvy**. This wide-spread, low-level condition is considered normal and eventually leads to sores (lesions) in the walls of blood vessels. **Vitamin C** is required to keep blood vessels strong and healthy. According to theory, the damage caused by *chronic scurvy* results from mechanical stress in the arteries, especially in regions where there is turbulence or high blood pressure.

Dr. Matthias Rath's analogy for the heart beat squeezing the coronary arteries is that of stepping on a garden hose roughly 80 times every minute.

Evolution has provided humans with a solution to problem of *chronic scurvy*, i.e., not enough **vitamin C** in the diet. The internal damage caused by chronic scurvy would eventually weaken blood vessels to the point of rupture, if not for one life saving factor:

ATHEROSCLEROTIC PLAQUE

A Nobel prize in Medicine was awarded for the finding that *lesions in the walls of blood vessels are the primary cause of atherosclerotic plaques.* Clusters of "fat" molecules acting as repair agents form nature's "plaster casts" against the weakness of blood vessel walls. These sticky molecules are lipoprotein(a). If these deposits continue to develop in the arteries of the heart they lead to

heart attack. If they continue to develop in the arteries of the brain they lead to stroke. Hundreds of investigators found that only a specific type of cholesterol molecule, **lipoprotein-(a)**, or **Lp(a)** for short, is the primary material that binds to a lesion in the walls of an artery forming plaques. **Lp(a)** is an ordinary LDL cholesterol molecule with a sticky apo-protein(a) (or **apo(a)**) attached to the surface. *Animals that make their own vitamin C generally do not have the variant **Lp(a)** cholesterol molecules.*

Pauling's revolutionary concept is that lipoprotein-(a) cholesterol in the blood is an evolutionary adaption for, and a *symptom* of, low vitamin C.

After crucial experiments, Dr. Pauling believed that the vitamin C/lysine protocol (as he presents on his video) would save lives by: a) Preventing chronic scurvy, b) Strengthening and healing blood vessels, c) Keeping Lp(a) blood levels low and d) Inhibiting the binding of Lp(a) molecules to blood vessel walls.

WHY DON'T ANIMALS GET HEART ATTACKS?

Can the absence of cardiovascular disease in animals confirm the Pauling/Rath Unified Theory? According to veterinary text books, *heart attack, stroke – cardiovascular disease in general -- is*

largely unknown in animals. Dr. Rath points out that if scientists could determine the reason, they would have an important clue to the puzzle.

Irwin Stone introduced Linus Pauling to the benefits of high amounts of **vitamin C**. Stone wrote, "*We can surmise that the production of ascorbic acid (vitamin C) was an early accomplishment of the life process because of its wide distribution in nearly all present-day living organisms. It is produced in comparatively large amounts in the simplest plants and the most complex; it is synthesized in the most primitive animal species as well as in the most highly organized. Except possibly for a few micro-organisms, those species of animals that cannot make their own ascorbic acid are the exceptions and require it in their food if they are to survive. Without it, life cannot exist. Because of its nearly universal presence in both plants and animals we can also assume that its production was well organized before the time when evolving life forms diverged along separate plant and animal."* [I. Stone, *THE HEALING FACTOR: Vitamin C Against Disease*, '76]

You might wonder, Don't animals die young? The average life span of the Elephant is 60 years. There are other species (e.g. Giant Tortoises) that can live over 100 years. Why don't most animals (other than primates, guinea pigs, fruit bats and a few parrots) suffer from heart disease? The reason, according to Linus Pauling:

Most animals produce their own vitamin C in amounts varying between 3,000 mg - 12,000 mg per day adjusted for body weight.

Most animals convert ordinary sugar (sucrose) to **vitamin C** in their kidneys or their liver. The newly created vitamin enters directly into the animal's blood stream. Animals also get some **vitamin C** from their food.

Humans cannot produce a single molecule of ***vitamin C*** *and must rely entirely on the diet. Tissue levels of* ***ascorbic acid****, the technical name for* ***vitamin C****, may be the most important difference between the physiology of primates (including humans) and other beings.*

It is an important finding that the few animal species that do *not* produce **vitamin C**, such as the primates and guinea pigs, *do* produce the "sticky" **Lp(a)** molecules.

Pauling recommended that people supplement their diet with **vitamin C** in amounts hundreds of times higher than the **60 mg** amount recommended by dietary authorities in the USA. *If animal meta-bolisms are any guide, Pauling's recommendations may be too low.*

Pauling's own experiments *showed that roughly 1/2 the amount of* ***vitamin C*** *ingested may be lost in the digestive tract or by urinary excretion.* [1986 Pauling video on Cancer]

BUT WHAT IF YOU ALREADY HAVE HEART DISEASE?

If the Pauling/Rath *Unified Theory* is correct, then the most likely reason you have heart disease is because your **vitamin C** intake has been less than optimal. *You will want to increase your **vitamin C**.*

What about existing plaques?

After investigators determined that **Lp(a)** causes atherosclerosis, the next question for scientists became: "*What causes these "fat" molecules to stick to the artery wall?" The Nobel prize winning answer turned out to be amino acid residues -- the so-called **Lysine** (and **Proline**) **Binding Sites** or **LBS.***

The **Lp(a)** or "cholesterol" binding sites are really just collagen protein (amino acid) residues that becomes exposed when blood vessels "crack." These exposed residues attract the **Lp(a)** molecules creating the plaque.

. . . A very important discovery. . .

What if the sticky **Lp(a)** molecules could be reduced, or were attracted some place other than the damaged arterial wall?

"Knowing that lysyl residues are what causes lipoprotein-(a) to get stuck to the wall of the artery and form plaques, any physical chemist would say at once that the thing to do is prevent that by putting the amino acid lysine in the blood to a greater extent than it is normally." [Linus Pauling, JON, Aug. '94]

Pauling's invention is to increase the lysine concentration in the blood serum causing Lp(a) to bind with lysine molecules in the blood rendering the Lp(a) inactive.

Today, there are more than 1100 MEDLINE (US National Medical Database) references to **Lp(a)**. These reports confirm the Nobel prize winning research paper of Brown-Goldstein and provide a solid foundation for the Pauling *Unified Theory*. The fundamental and now generally recognized scientific fact is that **Lp(a)** cholesterol molecules bind to blood vessel walls via the **Lysine** and **Proline** Binding Sites forming atherosclerotic plaques and occlusive cardiovascular disease.

A corollary is that ordinary LDL (so-called "bad") cholesterol is ***not*** the primary cause of plaque build-up, any more than calcium.

Massive research now supports the insight of Linus Pauling, who well ahead of his time in 1994 pointed out, *"If you have more than 20mg/dl of*

Lp(a) *in your blood it begins depositing plaques causing atherosclerosis."*

The Pauling/Rath U. S. Patent # 5,278,189 is for the ***prevention and treatment of occlusive cardiovascular disease with vitamin C and substances that inhibit the binding of lipoprotein-(a).*** The patent provides a method for the prevention and treatment of cardiovascular disease, such as atherosclerosis, by administering therapeutically effective dosages of a formula composed of **vitamin C**, lipoprotein-(a) binding inhibitors (e.g., **lysine** and **proline** or their analogs) and antioxidants.

Linus Pauling and Matthias Rath discovered that substances that inhibit the binding of lipoprotein-(a) also cause lipoprotein-(a) to be *released* from the arterial wall. According to the patent, a binding inhibitor (e.g., **lysine** or lysine analog) used alone or in conjunction with **vitamin C**, finds Lp(a) in the blood and binds with it before the molecule can reach the walls of arteries. *At high enough concentrations, the lysine in the blood attracts* ***Lp(a)*** *in the existing plaques and will dissolve the plaque.*

The inescapable conclusion is that the essential, very low cost and completely nontoxic amino acid lysine, when taken with vitamin C in amounts far larger than one

normally consumes in the diet, may reduce or may even eliminate Lp(a) based atherosclerosis in human beings.

The lysine treatment mechanism doesn't depend on the reason the Lp(a) based plaque forms. It really doesn't matter whether the arterial lesions were caused by mechanical stress, a vitamin deficiency, oxidized cholesterol, elevated homocysteine, fat in the diet, *or even little green men...*

HEART ATTACKS -- A LOOK BACK

The following historical perspective and commentary is adapted from various sources, including a published interview with Linus Pauling in the August 1994 Journal of Optimum Nutrition, books published by Earl Mindell and Drs. Eades, the Wayne Martin articles published in The Townsend Letter for Doctors and Patients, and Dr. Julian Whitaker's Health and Healing. It is provided here for informational purposes only and should in no way be considered an endorsement of any product or therapy by these publications or authors.

From the pen of Wayne Martin (January 1998):

This is to suggest that deaths from heart attacks and cancer can be reduced by half without changing our lifestyles all that much.

First let's address the subject of deaths from the kind of heart attack now called myocardial infarction (MI) but which 70 years ago was called coronary thrombosis.

There are in the USA now about 500,000 such deaths a year but this is a new disease that has come about in this century. Prior to 1925 there was almost no knowledge of this disease.

Beginning in 1975 Dr. Rodney Finlayson, who is now in retirement in North Devon in England, did a study of the records

of several London hospitals dating back to 1869 looking for case histories that were obvious cases of death from MI. Here is what he found. His figures are a ratio of deaths from MI per unit of population. From 1869 until 1900 there was one MI death per unit. By 1910 there were 10 MI deaths per unit. This was the time when machine made cigarettes were on the market and Dr. Finlayson suggested that cigarette smoking had caused deaths from MI to increase by a factor of ten. By 1980 MI deaths per unit of population had increased to 80.

Dr. Finlayson did not know it but at the same time Dr. Alaster Mackinnon had been doing the same study in Yorkshire. This study was of a constant population, of about 22,000. The Mackinnon results were the same as in the Finlayson study, that is that MI deaths increased in a ratio of one in 1900 to 10 in 1910 to 80 in 1980.

There were in the late 1960s reports of two populations in the world who were as free from MI deaths as was the English population in 1900. One such population was the black population in Uganda and the other was the population living near Udiapur in north India. This suggests that we could again be as free from MI deaths as was the population of England in 1900.

We will now go back to 1882 when Dr. Bizzozero discovered blood platelets and suggested that they start all blood clots. He had it all figured out right then that the beginning of all blood clots is a white or platelet thrombus. He said that in a blood clot there is first the platelet thrombus followed by the more massive fibrin thrombus. The problem was that he was about the only doctor in the world who could be made to believe this finding. However the editor of *The Lancet* in 1883 made favorable comment about Dr. Bizzozero's discovery. Nearly all doctors then were certain that there were no such things as blood platelets.

In 1926, Tait and Burk of the University of Montreal redid the work of Bizzozero and reconfirmed the nature of a platelet thrombus (blood clot). They had no better luck in convincing doctors than did Bizzozero. At this time, a blood clot was thought to be made of fibrin. Eventually the medical community recognized the existence of blood platelets, however, did not believe they were responsible for blood clots or disease states.

In 1930 Professor Karl Link of the University of Wisconsin discovered warfarin and believed it would prevent the formation of fibrin in a blood clot. After the discovery the university made millions of dollars on the sale of warfarin to kill rats; a massive dose would cause internal bleeding.

In 1945 doctors began to treat heart attack patients with a low dose of warfarin to prevent the formation of fibrin in coronary artery blood clots. Thereafter heart attack patients by the millions were maintained on a low dose of warfarin for years.

By 1955 the medical community concluded warfarin would not prevent a second heart attack, so cholesterol was discovered and said to be the cause of heart attacks. As blood clots seemed to no longer be the problem, the name of the disease was changed to myocardial infarction.

The Prudent Diet

Research on the polyunsaturated liquid vegetable fats, such as corn oil, in diet showed these fats would reduce cholesterol in blood slightly, which led to the birth of the Prudent Diet. With the Prudent Diet, the amount of polyunsaturated vegetable fats should double the amount of saturated animal fats.

It is difficult to believe but almost overnight cardiologists had unlimited faith in the Prudent Diet as being the ultimate answer to the pandemic of MI's. Having said that every one should live on the Prudent Diet to keep from having a heart attack, cardiologists then set out to prove this conjecture.

The first study was the Joliffe Anti-Coronary Club in New York City. Joliffe was a doctor working for the city. He was a diabetic and a vascular wreck. He was in a wheelchair and had gone blind in one eye. He looked to the Prudent Diet for his salvation.

The control group in this trial were men of wealth on Wall Street who could afford good food, such as eggs, butter, cheese and beef. The Prudent Diet group consisted mainly of the teaching staff at universities in the city. A pharmaceutical company made a special margarine, rich in polyunsaturated fats, to be used in the Prudent Diet group. The Prudent Diet was very strict in having almost no butterfat and very little red meat.

The trial ran four years and was hailed a great success because total serum cholesterol levels were reduced by 25 percentage points. One had to read the fine print, however, to discover that eight men on the Prudent Diet had fatal heart attacks, whereas none of the controls eating eggs, butter and beef had died. Then Dr. Joliffe died, it was said, of complications of his diabetic condition. He may have had a heart attack.

Cardiologists then decided that to prove the need for the Prudent Diet, there would have to be an anti-coronary club formed with over a million men enrolled. This was the National Diet Heart Study and was formed by Dr. Irwine Page of the Cleveland Clinic who had survived a heart attack. This trial had millions of government dollars to support it. There were food warehouses set up in seven cities where men on the Prudent Diet could get (free) the proper food among which were polyunsaturated donuts.

Before they would get one million men involved, there would first be a pretrial of two thousand men. This pretrial ran for three years and the results were an utter failure. There were exactly the same number of heart attacks, both fatal and non-fatal in both groups, the ones on the Prudent Diet and the controls. This trial had been started with the maximum of PR and fanfare; it was terminated with no public announcement for reasons of "cost".

I saw Dr. Page at the beginning of this trial and he was absolutely confident that the Prudent Diet was going to keep him from having another heart attack. He then died of a second heart attack.

Food for thought: In the Joliffe trial there were no MI deaths among the control group who were living on a diet very much like the diet of 1900 in the USA. In the National Diet Heart Study, there were the same number of fatal heart attacks in both the Prudent Diet and the control group. By this time the entire nation was living on something very close to the Prudent Diet. The men in the Prudent Diet group were living on a diet that was just a little more Prudent.

In England, during the same time, there was a study that had patients who had survived a heart attack and had similar results. Here again the Prudent Diet was an utter failure with the

same number of fatal heart attacks in both the Prudent Diet group and the controls.

Then there was an 8-year trial of the Prudent Diet at a VA hospital in California in which cancer deaths increased among the patients on the Prudent Diet by 15%. After this study there was an editorial in the *British Medical Journal* that suggested a relationship between the intake of polyunsaturated fats and the development of cancer.

By now we were up to 1970 and notwithstanding the failures of the diet trials, the Prudent Diet had become engraved in stone as the way to avoid having heart disease. Most of the population of our western world were living on something very close to the Prudent Diet.

The Prudent Diet Increases the Incidence of Heart Attacks

Five populations were studied, all of which suggested that the Prudent Diet was causing heart attacks. First there was the Roseto, Pennsylvania study. This small city is 100% Italian, prosperous Italians. *If the Prudent Diet was right, then what these Italians were doing was wrong.* They had a diet high in fats from cheese and meatballs cooked with olive oil, which is not very polyunsaturated. In 1955, among this Italian population, there were one-third the MI deaths as compared to other cities in eastern Pennsylvania and the nation as a whole. Here it was suggested that living in big families reduced the stresses of life thus preventing heart attacks. In no way could it be suggested that the Prudent Diet was causing deaths from heart disease. By 1970 the population of Roseto was having the same number of MI deaths as the nation, doctors having persuaded them to live on the Prudent Diet.

Next was the Boston Irish Brothers Study. Here brothers were studied, one remained in Ireland and one went to Boston. In 1965, the population of Ireland consumed large amounts of saturated animal fat in milk, butter, and meat and very little of the "good polyunsaturated fats." It was fully expected that the brothers in Boston would have fewer MI deaths than the brothers in Ireland. What they found was just the opposite, the butter-eating brothers in Ireland were having far fewer MI deaths.

Cardiologists said that men in Ireland did more walking that kept them from having the same amount of fatalities from heart disease. Again there could be no suggestion that the Prudent Diet was a factor in the group with increased MI deaths.

Then there was a study in India. The population in North India were found to be nearly free from MI deaths. These Indians in 1970 were the world's biggest eaters of a fat substance called ghee. They also consumed meat and large amounts of onions and garlic. At this time there was a strict vegetarian population in the south of India who lived on the Prudent Diet more closely than anyone in the USA. They had a high fat diet in which all fats were polyunsaturated vegetable oil or margarine made from it. They were having 15 times the MI deaths as compared to the butterfat-eating north Indians. By 1988, things had changed in the north of India. Low cost liquid polyunsaturated vegetable oil had priced ghee out of the market. Also, doctors were teaching the Prudent Diet. By then the death rate from MI in north India had increased to match that in the USA.

A significant finding came from a study of the French and Scottish populations. Their diets were similar with the French consuming more butter and cheese. Both populations had elevated serum cholesterol levels, however the mortality rate from MI in Scotland was five times greater among men and eleven times greater among women. The difference was that the French were drinking about 400 cc. a day of wine. The grapes are rich with bioflavonoids and antioxidants.

Polyunsaturated Fatty Acids Increase the Incidence of Heart Disease

I have had a long friendship with Professor Terrence Anderson who was the administrator of the School of Public Health at the University of British Columbia. He maintains that the pandemic of MI deaths since 1900 has been caused by consuming large amounts of polyunsaturated vegetable fats from which the antioxidants have been removed. It is a fact that as MI deaths have increased 80-fold since 1900, polyunsaturated fats have increased in diet by a factor of three. The population studies also correlate diets high in polyunsaturated fatty acids with increasing MI deaths.

Just look at the fats in diet in 1900 when MI was unknown and most rare. They were butter and lard. The only polyunsaturated vegetable oil to be had then was cottonseed oil and the use of it was minuscule.

In 1980 cardiologists resurrected platelets and blood clots as a cause of MI deaths - and told everyone over 40 to take aspirin to prevent having a heart attack. One factor in the prevention of MI is the adhesiveness of platelets as the greater the adhesion of platelets the greater the chance of having a coronary blood clot.

Then came a series of trials on aspirin in the prevention of MI. The results were about the same as Prudent Diet trials. There were in the 1970s two trials in England that were failures. No benefit or very slight benefit was found for aspirin in the prevention of MI. This was followed by a much larger US government-financed trial in the USA and reported in 1980. This trial was an abject failure with much bleeding of the stomach due to aspirin and no benefit at all in the prevention of MI.

Doctors felt that the case could be made for aspirin if only doctors were the subjects. A trial in England among doctors was again a failure, however a larger trial among doctors in the USA was hailed as a great success. In this American trial, non-fatal heart attacks were reduced by 40%. The bad news however, was that fatal heart attacks were not reduced and moreover overall survival was not increased. Nonetheless as the result of this trial, it was suggested or even demanded that all men over 40 should be taking aspirin.

There was something a bit different about this trial among doctors in the USA. Bufferin was used and Bufferin contains both aspirin and some magnesium. Magnesium is greatly beneficial to the heart. It reduces platelet adhesion, is a vasodilator and is a potent antiarrhythmic agent.

Platelet Adhesiveness

At the National Heart Hospital in London circa 1970, they were using a test for platelet adhesion and the results were stated as PAI, platelet adhesiveness index. In this test a blood sample was taken and a platelet count was made. Then a second blood sample was taken and this time the blood was passed over glass beads. If half the platelets stuck to the

beads, PAI was 50. Patients who had survived a heart attack would have PAI of 50 and hence were considered to be at risk of death from a second heart attack. Young women who never suffer from MI have PAI of 20 yet they will have proper blood clots in wounds.

At the National Heart Hospital, in the years 1960 to 1965, they did a PAI test on every patient to come to this hospital and they never found a single patient with PAI less than 40. They felt anyone with a PAI of less than 40 was not going to have a heart attack. Put another way, they felt that the great problem about MI was one of blood clots in coronary arteries.

The idea of testing for PAI never came to the USA.

There are all kinds of things other than aspirin that reduce PAI, one of which is the drug dipyridamole. Here mention will be made of the European Stroke Prevention Study. About 90% of strokes are thrombotic strokes, blood clots in blood vessels in the brain. This trial had as subjects patients who had had an indication of a stroke. First aspirin alone was used with little or no benefit. Then dipyridamole was added to treatment, 300 mg a day and the results were outstanding. Stroke deaths were reduced by 50%, heart attack deaths by 35% and cancer deaths by 25%.

There are many things that reduce PAI better than aspirin. Vitamin E at 400 iu a day will, as will Vitamin B6 at over 40 mg day. There was an editorial in *The Lancet* a few years ago on how anti-thrombic is vitamin B6 at over 40 Mg. So is fish oil. This is the omega-3 fatty acid that we have been hearing so much about of late. Then recently from the University of Wisconsin has been a report that purple grape juice at 10 oz. a day will reduce PAI better than aspirin. It has been suggested that gamma linolenic acid in evening primrose oil will reduce PAI better than anything else. Also the oils of onion and garlic will reduce PAI. Ground ginger also is greatly effective in reducing PAI and like aspirin, it will reduce pain. It is highly anti-inflammatory. It is a sad state of affairs that doctors in the USA have gotten most men over 40 taking aspirin while not setting up a test to see if it is in fact, reducing PAI.

Antioxidants Prevent MI Deaths

Now back to the teaching of Terrence Anderson. He says that antioxidants are greatly important in the prevention of MI deaths. The 10-year Zutphen study has just been reported. This is a study of a small city in the Netherlands that started out to find out about cholesterol and MI deaths. After five years, there was no good association in this study between cholesterol and MI deaths so it was changed to be a study of one antioxidant in diet, quercitin. This is a flavonoid antioxidant. In this population, quercitin came from black tea and onions. The part of this population with less than 10 mg a day of quercitin were having almost double the death rate from MI as compared to the ones with over 30 mg of quercitin. There were 39 MI deaths among the men with less than 10 mg of quercitin and only 21 among the men with the highest quercitin in diet. I say why bother with 30 mg of quercitin a day. I take 1,000 mg.

Whole grains contain antioxidants, mostly tocopherols. Vitamin E is alpha tocopherol. Now back to the teaching of Anderson, he says that after 1910 in our Western World, there was a great loss of tocopherol antioxidants as wheat flour was made white by bleaching with strong oxidizing agents which abolished the tocopherols. Deaths from MI began to increase as white bread replaced whole grain bread. He cites the case of Italy. Beginning in 1919 and until 1946, Italy banned the milling of white flour, this to reduce the importation of wheat. During this period all bread was whole grain. The big increase in MI deaths that occurred in the USA and England did not happen until after 1946 In Italy.

Anderson attributes the lesser death rate from MI among the brothers in Ireland in the Boston Irish Brother Study to the very large consumption of oatmeal porridge in Ireland, a whole grain rich in tocopherols.

We now have many new antioxidants available to us. One is coenzyme Q10 which now has worldwide usage in treating congestive heart disease. There is melatonin which like coenzyme Q10, we make a little of. About 25 years ago, cardiologists were much concerned about catecholamines causing heart attacks. If a man is exposed to great stress and excitement such as running to a fire, catecholamines are released in the myocardium causing severe damage to the heart. They most likely set off fatal arrhythmias which are followed by coronary blood clots.

In a recent letter to *The Lancet* there was a report that melatonin inhibits catecholamine release. In this study, patients who had survived a heart attack had low melatonin in blood as compared to men who had not had a heart attack.

Two more antioxidants that are now available in pills are N acetyl cysteine and alpha lipoic acid.

Vitamin A is a weak antioxidant, but there was a report in *The Lancet* a few years ago from Belgium showing that a high level of vitamin A in blood is protective against thrombotic strokes. Here it was explained that when a blood clot in a vessel in the brain shuts off oxygen to the brain nearby, free radical action begins to kill brain cells. If there is enough vitamin A in blood and tissue, this free radical killing of brain cells will not happen. The body has the ability to lyse small blood clots and in time to restore oxygen to the brain. A high level of vitamin A in blood and tissue will give the natural fibrinolytic action time to happen, fibrinolytic meaning the lysing of blood clots.

Lately, there have been super potent antioxidants found in grape seed and in the skin of grapes.

LDL Cholesterol Only Bad When Oxidized

At the end of 1996 there was a year end supplement on heart attacks in *The Lancet*. By now it is known that the major bulk of human atheroma is not fat or cholesterol but rather is mostly made up of proliferating smooth muscle cells. There is however, an effect that cholesterol has on causing heart attack. For many years it has been known that we have two types of cholesterol, LDL cholesterol which is the bad form and HDL cholesterol which is said to be good cholesterol. Now it is understood that LDL cholesterol when oxidized, has the effect of causing spasms of vasoconstriction of muscles in coronary arteries and that these spasms then start a heart attack by causing arrythmias. If one has a proper amount of antioxidants in diet, then LDL cholesterol will not be oxidized. It could be that cholesterol is only harmful when LDL cholesterol is oxidized.

The population of Okinawa has a very low death rate from MI while having serum cholesterol that is high by current orthodox standards. What is shaping up is that if a person has in diet enough antioxidants to prevent the oxidation of LDL

cholesterol, then a moderate elevation in blood of cholesterol may not be causing heart attacks at all.

Circa 1950, Dr. Henry Schroeder of Dartmouth Medical College had done a study in the USA on hard vs soft water and he found far fewer heart attack deaths in areas of very hard water. He concentrated his studies on two cities, Lincoln, Nebraska and Savannah, Georgia. Lincoln had water with a hardness of 200 ppm. Savannah had water With 55 ppm. Savannah was having over two times the heart attack death rate as compared to Lincoln. Water softeners came into almost universal usage after 1940. They no doubt played a part in increasing deaths from MI from 1 in 1900 to 80 in 1980. Dr. Schroeder felt that if that part of the population of Lincoln who had water softeners were factored out, then the population of Savannah would be having three times the death rate from heart attacks. Dr. Schroeder was unable to finish this study due to his developing cancer. Dr. Schroder did not know it in 1950, but the great benefit due to hard water is the magnesium connection. Here is an interesting thought: The hard water in Lincoln, Nebraska and French wine have the same magnesium content. I buy liquid magnesium citrate and drink about one ounce a day.

I think that Anderson was right in saying that a big increase in polyunsaturated fats in diet from which the antioxidants have been largely removed, has been causing the big increase in MI deaths. The question then is what would be the effect of these polyunsaturated fats along with a big packet of antioxidants. No one knows for there has been no studies of this combination.

In about 1970 I visited Dr. Kurt Oster, cardiologist at Fairfield University. Homogenized milk came on the market in 1936 and Dr. Oster holds that it has greatly increased deaths from MI. In any event, it came along at a time when MI deaths were increasing at a rapid rate. The cow puts xanthine oxidase, an enzyme that causes oxidation, in milk in big particles which are excreted in feces. When milk is homogenized, the xanthine oxidase particles are made very small and they pass right into the blood Circulation. He claims that that this enzyme causes the vasospams that precede the coronary blood clots of MI.

In 1981 Dr. Stephen Seely of the University of Manchester published a report on liquid milk consumption vs MI deaths. He compared Finland with 30 grams of protein a day

from liquid milk, to Germany with 14 grams to Yugoslavia with 8 grams to Japan with 2.5 grams. Germany had only half the MI death rate of Finland. Yugoslavia had one fourth and Japan only one-tenth, the death rate from MI as compared to Finland.

Seely had started this study with the idea that butterfat may be causing MI deaths and his liquid milk study supported this concept, however he had a problem. He had done a worldwide study on cheeses and found that there is not the least association between cheese consumption and MI deaths. The xanthine oxidase concept was most welcome to him. Milk is not homogenized before it is made into, cheese.

Finally, in 1966 and 1967 Boris Sokoloff, et. al. published their findings about vitamin C and cardiovascular disease. When blood is high in tryglycerides, it is turbid. Then the tryglycerides are acted on by lipoprotein lapase (LPL) made by the pancreas and the tryglycerides are converted to water soluble lipoproteins.

First with rabbits and then with humans, he showed that one to three grams a day of vitamin C would increase LPL production to produce more alpha lipoprotein while reducing both tryglycerides and the "bad" cholesterol.

Then Sokoloff got a group of 60 patients who were past aged 60 who had survived a thrombotic stroke or a heart attack. They all had elevated cholesterol and triglycerides and low production of LPL. They were all maintained on one to three grams a day of vitamin C for one year. There was not a single vascular event, heart attack or stroke in this group in that year when it was expected that there would be between six and 12 heart attacks or strokes in such a group.

I feel certain that without changing lifestyles much, we can all get back to where MI deaths can be as rare as they were in 1900. To do this, the first thing that would have to be stopped would be the Prudent Diet.

First Published in the Townsend Letter for Doctors and Patients
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Heart Surgery

"... the heart surgery industry is a fraud."
-- Julian Whitaker, MD

"The heart surgery industry is booming. According to American Heart Association statistics, in *1995* 1,460,000 angiograms (the diagnostic procedure that starts the ball rolling) were performed at an average cost of $10,880 per procedure. This resulted in 573,000 bypass surgeries at $44,820 a shot, and 419,000 angioplasties (the balloon procedure for opening up arteries) at $20,370 each. The total bill for these procedures is over $50 billion a year.

"The millions of people told that they needed immediate surgery to save their lives actually had a 98.4% chance of surviving without surgery!" [CASE study published by the *New England Journal of Med.*, 1983]

"There is no scientific justification for the use of angiography, balloon angioplasty and bypass surgery to treat most cardiovascular disease. A good example of this is angioplasty, in which a balloon on the tip of a catheter is used to open blockages. In my opinion, there is never a reason for anyone to have an angioplasty. It is a dangerous procedure looking desperately for validation. Whenever it is compared to a non surgical therapy -- and there have been very few of these studies -- patients treated with angioplasty virtually always fare worse. There is a higher death rate, higher heart attack rate and, in general, a higher repeat surgery rate. This procedure will, in my opinion, always be an unproven, expensive and dangerous gimmick that became an accepted therapy based on self-serving "presumption" only.

"Several studies over the past two decades, involving over 6,000 patients with heart disease, have shown that patients funneled into surgical procedures to do significantly worse than those treated with noninvasive techniques. "Noninvasive" refers to the use of medication, not diet, exercise and multiple vitamin supplements, which can be quite beneficial." Bypass surgery may be helpful for some patients, but it should not be used as the first treatment, and clearly not in mild heart

attack patients. Medication, dietary and lifestyle changes, plus nutritional supplements are more effective approaches"

"Other than their cost, the only thing definitely known about these procedures is that they do kill people. Roughly one in 25 patients having a bypass and about one in 65 undergoing angioplasty die from the procedure. Frankly, if we took all of the bypass surgeons and catheter-pushing cardiologists, tied their thumbs together and locked them in a closet, we would save close to 30,000 lives and over $50 billion ever year." *Julian Whitaker, Health & Healing, Sep 98, Vol 8, No. 9*

Cholesterol

Cholesterol: Villain or Hero?

This excerpt, from the informative book by Michael and Mary Dan Eades, MDs, PROTEIN POWER, offer an excellent overview of the life essential properties of cholesterol.

"The consensus on cholesterol seems to be the lower, the better, but as we shall see, this is not always the case. Far from being a health destroyer, cholesterol is absolutely essential for life. Although most people think of it as being "fat in the blood," only 7 percent of the body's cholesterol is found there. In fact, cholesterol is not really fat at all; it's a pearly-colored, waxy, solid alcohol that is soapy to the touch. The bulk of the cholesterol in your body, the other 93 percent, is located in every cell of the body, where its unique waxy, soapy consistency provides the cell membranes with their structural integrity and regulates the flow of nutrients into and waste products out of the cells.

In addition, among its other diverse and essential functions are these:

- Cholesterol is the building block from which you body makes several important hormones: the adrenal hormones (aldosterone, which helps regulate blood pressure, and hydrocortisone, the body's natural steroid) and the sex hormones (estrogen and testosterone). If you don't have enough cholesterol, you won't make enough sex hormones.
- Cholesterol is the main component of bile acids, which aid in the digestion of foods, particularly fatty foods. Without cholesterol we could not absorb the essential fat-soluble vitamins A, D, E and K from the food we eat.
- Cholesterol is necessary for normal growth and development of the brain and nervous system. Cholesterol coats the nerves and makes the transmission of nerve impulses possible.
- Cholesterol gives skin its ability to shed water.
- Cholesterol is a precursor of vitamin D in the skin. When exposed to sunlight, this precursor molecule is converted to is active form for use in the body.
- Cholesterol is important for normal growth and repair of tissues since every cell membrane and the organelles (the tiny structures inside the cells that carry out specific functions) within the cells are rich in cholesterol. For this reason newborn animals feed on milk or other cholesterol-rich foods, such as the yolks of eggs, which are there to provide food for the developing bird or chick embryos.
- Cholesterol plays a major role in the transportation of triglycerides -- blood fats -- through the circulatory system.

A quick review of this list should give you a better idea of what cholesterol does and dispel any notion that it is a destroyer of health to be feared and avoided at all costs. Far from being a serial killer, cholesterol is absolutely essential for good health; without it you would die. Without cholesterol we would lose the strength and stability of our

cells, rendering them much less resistant to invasion by infection and malignancy. In fact, a grave sign of serious illness, such as cancer development or crippling arthritis, is a falling cholesterol level."

Read more in PROTEIN POWER, Miachael R. Eades, MD, and Mary Dan Eades, MD, Bantam Books, 1996.

Pharmacist Earl Mindell on Cholesterol Lowering Drugs

Excerpts From:
Prescription Alternatives
by Earl Mindell, RPh, PhD
Virginia Hopkins, M. A.
Keats Publishing Inc. 1998

Debunking the Cholesterol Myths

". . . a victim of negative press, cholesterol is an essential component of the production of the steroid hormones and in nerve function as well as other essential body processes. First, there is absolutely no evidence anywhere that normal cholesterol floating around in the blood does any harm. In fact, cholesterol is the building block for all your steroid hormones, which includes all the sex hormones and the cortisones. Even slightly low levels of cholesterol are associated with depression, suicide, and lung cancer in older women. . . For most people, eating high cholesterol foods does not raise cholesterol...

Why Does Cholesterol Accumulate in the Arteries?

"Drug company advertising for cholesterol lowering drugs gives the impression that excessive cholesterol in the blood simply deposits on the artery wall, and that lowering cholesterol levels stops that process. . . High cholesterol is a symptom of an underlying nutritional deficiency and/or toxicity that damages the arteries. . .

The Myth of the Cholesterol Count

"Another cholesterol myth perpetuated by the drug companies is that everyone with a total cholesterol count over 200 mg/dl should be concerned. This is blatantly false. . .

Drugs to More Harm than Good

"While a cholesterol-lowering drug will usually do a very good job of lowering your cholesterol, there's scant, if any, evidence that it will help you live longer or reduce your risk of heart attack unless you are extremely ill or have just suffered from a heart attack. . . There are no studies that show women benefit from these drugs -- all the studies showing even marginal benefits have been done on men. Nor are there any studies showing that they reduce heart attacks or death in men aged 65 to 75. Since heart disease takes decades to develop, it's highly unlikely that cholesterol-lowering drugs will help anyone over the age of 75. That leaves men aged 35 to 55, but even here the evidence of benefit is slim, and the possible side effects are huge. . .

"If the American public had even a clue of how destructive these drugs are, they wouldn't touch them except in an emergency. . .

"Every information sheet on the most commonly prescribed cholesterol-lowering drugs will tell you that they cause cancer in rodents when taken long term in relatively normal doses. It's also well-known that they can cause severe emotional imbalances in men, along with a wide array of life-threatening side effects. . . The wisest course of action is to avoid these drugs... "

--Earl Mindell

WARNING

SO-CALLED "ULTRA FAST" HEART SCANS SCORE FALSE POSITIVES AS WELL AS FALSE NEGATIVES!

Today we are subjected to much advertising about a "painless" non-invasive procedure to test for atherosclerotic

build-ups, variously called "ultra fast heart scans." These expensive tests can detect the presence of calcium, which may or may not be an indicator of serious arterial blockage. It is well established that arteries can be clogged and blocked without calcium. In this case, the heart scan will report a **false negative**. In a recent case a former heart patient on the *Pauling Therapy* for more than two years reported undergoing such a test and having scored "massive heart disease." An invasive angiogram found very little plaque meaning the scan report was a **false positive**. According to this gentleman:

> *"The angiogram showed a couple of spots where the plaque was still there, nothing over 40% to 50% occluded. But there were long stretches of arteries, in fact most of the "left anterior descending" that the cardiologist referred to as "beautiful". This same stretch of artery was shown as dangerously occluded in the scan. In fact, so frightening that it caused me to have the angiogram."*

THE IMPORTANCE OF VITAMIN C

You may find it difficult to believe that the answer to the riddle of heart disease -- the leading cause of premature death in human beings -- might be something as simple as a **vitamin C** deficiency. You might be surprised.

Vitamin C and Heart Disease A Chronology

- 1941 Canadian cardiologist J. C. Patterson reports that more than 80% of his heart disease patients have low vitamin C levels.
- 1954 G. C. Willis shows that vitamin C supplementation can reduce arterial deposits.
- 1960s Boris Sokoloff gives 60 humans with CVD multi-gram vitamin C. No vascular events during year long study when 6-12 were expected .

- 1970 Linus Pauling publishes his first book on vitamin C.
- 1970s Vitamin C consumption in the U.S. rises by 300%. Mortality from heart disease decreases by 30% in the U.S.-the only country with a significant drop in heart disease fatalities.
- 1986 Pauling summarizes the evidence for vitamin C against heart disease and other diseases in his book "*How to Live Longer and Feel Better*", which becomes a best-seller.
- 1989 Rath and Pauling discover that optimum dietary vitamin C prevents the deposition of lipoprotein(a) in artery walls.
- 1991 Rath and Pauling publish *"Solution to the Puzzle of Human Cardiovascular Disease*".
- 1992 Dr. Enstrom and colleagues (UCLA) show, in over 11,000 Americans, that increased intake of vitamin C reduces the death rate from heart disease by nearly half and prolongs life for more than six years.
- 1993 Pauling films Unified Theory of Heart Disease: Cause and Cure video.
- 1997 More than 1100 MEDLINE references to lipoprotein(a) since 1994, but none test Pauling's claim of a possible vitamin C/lysine cure.
- 1998 The Vitamin C Foundation submits study proposal to National Institutes of Health to formally investigate Pauling's claim.

SUMMARY OF THE RESEARCH

"There are more than ten thousand published scientific papers that make it quite clear that there is not one body process (such as what goes on inside cells or tissues) and not one disease or syndrome (from the common cold to leprosy) that is not influenced -- directly or indirectly -- by vitamin C." [Dr. Emanuel Cheraskin, Dr. Ringsdorf and Dr. Sisley from **THE VITAMIN C CONNECTION, 1986]**

In 1941 Canadian cardiologist. J. C. Paterson reports that more than 80% of his heart disease patients have low **vitamin C** levels compared to controls. In 1954 Willis shows that **vitamin C** supplementation can reduce arterial deposits, first in guinea pigs and then in humans using a serial x-ray technique.

Guinea pig experiments are repeatable, these animals share with man the inability to make **vitamin C**, and show an association between low ascorbate and atherosclerosis, and recently **Lp(a)**. Two of these guinea pig studies were followed up with human prompting the Canadian Dr. Willis, to declare that atherosclerosis is reversible.

During the 1960s Biochemist Irwin Stone and others recommend increased dietary **vitamin C** supplementation to improve health. In 1966 Dr. Boris Sokoloff and associates study **vitamin C** in relation to CVD, first in guinea pigs, and later 60 patients who were past aged 60 who had survived a thrombotic stroke or heart attack. All were maintained on one to three grams a day of **vitamin C** for one year. In 50 out of 60, cardiovascular indications improved. There was not a single vascular event, heart attack or stroke in this group that year when between six and 12 heart attacks or strokes were expected.

In 1970 Dr. Linus Pauling publishes his first book on **vitamin C**. During the 1970s his institute reports that **vitamin C** consumption in the U.S. rises by 300%. Mortality from heart disease decreases by 30% in the U.S.-the only country with a significant drop in heart disease fatalities.

Ginter and Schafer publish their studies on the connection between **vitamin C** and atherosclerosis. In 1986 Dr. Pauling summarizes the evidence for **vitamin C** against heart disease and other diseases in his book *"How to Live Longer and Feel Better"*, which becomes a best-seller and has an extensive bibliography.

In 1989 Dr. Rath and Dr. Pauling discover that optimum dietary **vitamin C** prevents the deposition of

lipoprotein(a) in artery walls. In 1991 Dr. Rath and Dr. Pauling publish "*Solution to the Puzzle of Human Cardiovascular Disease*". This paper explains (a) that **vitamin C** deficiency is the direct and most frequent cause of heart attacks, (b) how plasma risk factors lead to atherosclerotic deposits in arterial walls, (c) why humans suffer from heart attack and stroke but rarely from failure of other organs, and (d) why animal species who are able to produce their own **vitamin C** in the body do not develop heart disease.

In 1992 Dr. Enstrom and colleagues (UCLA) show, in over 11,000 Americans, that increased intake of **vitamin C** reduces the death rate from heart disease by nearly half and prolongs life for more than six years. (The majority of the 1100 Medline Lp(a) studies are from 1992 forward.)

A 1997 Japanese study published in Circulation correlates **Lp(a)** to restenosis (re-occurrence of plaque). A multivariate-analysis revealed that the absolute change in **Lp(a)** (before versus 1 day after PTCA) to be the sole significant predictor of restenosis among the clinical, angiographic, and plasma lipid parameters examined. The conclusion was that **Lp(a)** levels were significantly higher in the restenosis group, and they fell significantly after PTCA in the restenosis group.

In 1997 Stubbs, et. al. associated **Lp(a)** with unstable angina. This study provides the first evidence in man of a significant role for lipoprotein(a) in unstable angina ($P = 0.0001$).

A study of 1,605 randomly selected man in Finland, aged 42 to 60 years, was conducted between 1984 and 1989. None of the men had evidence of pre-existing heart disease. After adjusting for other confounding factors, men who were deficient in **vitamin C** had 3.5 times more heart attacks than men who were not deficient in **vitamin C**. The scientists' conclusion was, "*Vitamin C deficiency, as assessed by low plasma ascorbate concentration, is a risk*

factor for coronary heart disease." British Medical Journal Vol 314, Iss 708, 1997.

In 1996, researchers at the University of Chicago discovered the **Lp(a) proline** binding site. In private correspondence one study author wrote:

"Our study published in JCI demonstrated for the first time that L-proline can inhibit the binding of Lp(a) and apo(a) to fibrinogen in vitro conditions. However, we have no data about the physiological relevance of this finding. It is well established that the binding of Lp(a) to fibrinogen also involves the lysine binding sites of apo(a) (component of Lp(a)), therefore, the presence of lysine and lysine analogue dramatically reduce this binding. Once again, the in vivo relevance of this inhibition is unknown.

Sincerely, Olga Klezovitch, Ph.D. University of Chicago

SERIAL ARTERIOGRAPHY IN ATHEROSCLEROSIS (in Human Beings)

G. C. Willis, MD; A. W. Light, MD; W. S. Gow, MD

Canad. M. A. J.; Dec 1954, Vol 71

[From the paper] The patients studies by arteriography were selected from Queen Mary Veterans' and St. Anne's Hospitals. All were men, varying in age from 55 to 77, with an average age of 64 years, and were those who had shown many of the clinical manifestations ordinarily considered to be associated with atherosclerosis. Bilateral femoral arteriography was performed in all cases. [This technique is described in the paper along with remarkable pictures of plaque in human arteries.]

*Controls

# / age	*Diagnosis	*Time observed in Days	*Change in Plaques	*Cholesterol **before** mg/%	*Cholesterol **after** mg/%	*Symptom Changes
1/ 72	Severe peripheral atherosclerosis	176	2 plaques **bigger**, 2 unchanged	N/A	N/A	Impending gangrene
2/ 74	Severe peripheral atherosclerosis	70	4 plaques **bigger**; multiple small plaques unchanged	332 mg/%	290 mg/%	No Change

3/ 63	Diabetes	70	**No Change**	240 mg/%	278 mg/%	No Change
4/ 77	Atherosclerotic Heart Disease, diabetes	82	**No Change**	216 %/mg	232 mg/%	No Chnage
5/ 59	Severe periph. atherosclerosis	172	1 plaque **bigger**	N/A	N/A	Required amputatio n
6/ 62	Diabetes	192	**No Change**	332 mg/%	287 mg/%	No Change

None of the patients, with the exception of diabetics, was given any special diet. The treated group were given 500 mg of ascorbic acid (vitamin C) orally (three times a day) but otherwise were the same as the control group.

*

*Results in Group Given Ascorbic Acid (500 mg)

* # / age	*Diagnosis	*Time observe d in Days	*Change in Plaques	* Cholest erol **before** mg/%	* Chole sterol **after** mg/%	*Symptom Changes
7/ 69	Severe atherosclerotic heart disease	62 days	3 plaques **bigger**, 2 unchanged	350 mg/%	262 mg/%	Died 1 month later of pneumoni a
8*/ 59	Severe periph. atherosclerosis Amp. left.	172 days	2 plaques **smaller**	375 mg/%	360 mg/%	No Change
9/ 72	Periph atherosclerosis, Imp. gangrene	136 days	3 plaques **smaller**, 3 unchanged	N/A	N/A	Claudicati on decreased
10* /58	Old myocardial infarction	125 days	2 plaques **bigger**, several unchanged	323 mg/%	287 mg/%	No Changed
11/ 56	Diabetes, Xanthomatosis. Ang. pectoris	110 days	7 plaques **smaller**, 7 unchanged	312 %mg	216,2 40,24 0 %/mg	Xanthoma ta softer and less painful but same size
12* /64	Hypercholesterolae mia, Old myocardial infarction	105 days	6 plaques **bigger**, 2 unchanged	560to4 35 %/mg	485 %/mg	No change
13/ 65	Old myocardial inf. Cerebral thrombosis	116 days	5 plaques **unchanged**	258 %/mg	255 %/mg	No Change
14/ 61	Diabetes. Old myocardial inf.	96 days	1 plaque **smaller**,	255 %/mg	312	No Change

			multiple unchanged			
15* /55	Diabetes	100 days	1 plaque **smaller**, 6 unchanged	221 %/mg	248 %/mg	No Change
16/ 63	Angina pectoris	155 days	3 plaques **smaller**, multiple unchanged	292 %/mg	390 %/mg	Angina much less

Notes: * These cases in error each had a period up to 3 weeks without therapy. All others had continuous therapy.

Conclusions

1. The problem and the importance of study atherosclerosis objectively during life are outlined and serial arteriography is suggested as a method of study.
2. Both progression and regression of plaques are observed to occur over relatively short periods of time.
3. Progression and regression did not co-exist in the same cases during one period of ' observation.

Preliminary results of ascorbic acid (Vitamin C) therapy in human atherosclerosis are encouraging. No plaques were found to be smaller in the controls. More than 60% in vit C group showed smaller plaque deposits.

THE REVERSIBILITY OF ATHEROSCLEROSIS
G. C Willis
Canad. M A. J., July 15, 1957, vol. 77

A total of 77 male and female adult guinea-pigs was rendered scorbutic... After intervals of from 21 to 30 days, 50 of these animals were given ascorbic acid therapy and there remaining 27 were sacrificed.

*The Number of Animals in the Various Experimental Groups, With and Without Atherosclerosis and the Degree of the lesions

*Experiment	* Total Animals	* With athero-sclerosis	* Without athero-sclerosis	*Average degree of athero-sclerosis
Scorbutogenic diet 42 days with **ascorbic acid added** from the beginning	12	0	12	0
Scorbutogenic diet for periods of from	27	11	16	2.5+

21 to 30 days				
Scorbutogenic diet for 21 to 30 days, then ascorbic acid for 1 to 5 days	25	9	16	2.5+
Scorbutogenic diet for 21 to 30 days, then ascorbic acid for 7 to 27 days	25	7	18	2.5+

Conclusions

No atherosclerosis was found in the controls (with ascorbic acid added from the beginning.) From the paper, When ascorbic acid is given to scorbutic (missing vitamin C) guinea-pigs, the early atherosclerotic lesions resorb quickly. The advanced lesions are considerably more resistant to reversal, apparently because of the islands of lipid whose only contact with the resorbing process is at the surface. A correlation is made between the atherosclerosis of the scorbutic guinea-pig and that observed in man, and the results of a previous study of ascorbic acid therapy in human atherosclerosis."

IF VITAMIN C REALLY IS THE ANSWER, WHY IS IT BEING IGNORED?

The history of science is the history of major developments that were not immediately recognized for their importance by the scientific community.

The Pauling *Unified Theory* of heart disease holds that humans, having lost the ability to manufacture their own **vitamin C** through a genetic defect, must obtain large amounts of this important substance in their diet or face the **Lp(a)** adaption - plaques. This theory conflicts with the hypothesis that dietary **vitamin C** is only necessary in small amounts to avoid frank clinical scurvy.

Unlike many other vitamins that act as co-enzymes to facilitate metabolic reactions in the body, **vitamin C** molecules are not only required -- they are transformed --

during the production of the protein collagen in the body. *Vitamin C is used up.* Pauling and his associate and heart researcher Dr. Matthias Rath, MD, independently formulated their own assessment regarding the cause and nature of occlusive heart disease. They agree that heart disease is essentially a repair process that is started by a lesion; their theories explain the primary reason these lesions occur. *Accordingly, occlusive cardiovascular disease is a natural body healing process for the walls of arteries and veins weak from insufficient collagen, a condition caused by inadequate vitamin C in the diet.*

These scientists performed careful laboratory experiments with guinea pigs in order to test their theory. They repeated earlier experiments and induced atherosclerosis in guinea pigs by restricting the intake of **vitamin C** to the U. S. RDA (adjusting for body weight).

The result of these experiments is always the same. Over time, roughly five weeks in guinea pigs, **vitamin C** restriction ultimately causes the animal's blood vessels to lose stability causing lesions that result in the arterial healing process we call atherosclerosis. Microscopic pictures show the animal lesions mimic the human form of the disease. **Lp(a)** levels in animals deprived of **vitamin C** were shown to rise as described in the Pauling/Rath 1994 U. S. Patent. Animals in the control group that were fed the same except for the addition of a human equivalent of 3 to 5 gm of **vitamin C** per day showed no signs of atherosclerosis. **Lp(a)** remained low in these animals.

This repeatable experiment proves beyond a reasonable doubt that a single factor restricted vitamin C in the diet causes atherosclerosis.

The subject of intense scientific scrutiny, this insight about the "missing link" in heart disease -- the coagulating

healing factor lipoprotein(a) -- has been confirmed during a reevaluation of The Framingham Heart Study.

According to Dr. Rath, studies show that special diet does not influence lipoprotein(a) blood levels. **Vitamin C** and vitamin B3 (niacin) can lower blood levels of lipoprotein(a).

Furthermore, according to Dr. Rath, significant documentation attests that:

"*Vitamins belong to the most powerful agents in the fight against heart disease. This fact has been established by studies of thousands of people over many years. Here are some important results of recent clinical studies:*

• *Vitamin C cuts heart disease rate almost in half (documented in 11,000 Americans over ten years)*

• *Vitamin E cuts heart disease rate by more than one third (documented in 36,000 Americans over six years.)*

• *Beta Carotene (provitamin A) cuts heart disease rate almost in half (documented in 36,000 Americans).*

• *No prescription drug has ever been shown to help prevent heart disease similar to [vitamins A, C and E]. These results and those of countless other studies are so clear that anybody questioning the value of vitamins in the prevention of heart disease may safely be considered as uninformed.*"

A Surprisingly Safe, Effective, and Simple Therapy?

Armed with the knowledge why plaque forms in human arteries, Linus Pauling invented an orthomolecular therapy for inhibiting the binding of **Lp(a)**:

The Pauling Therapy

lysine	**Vitamin C**
3 - 6 gm	3 - 6 gm
(3,000 - 6,000 mg)	(3,000 - 6000 mg)

Quoting Pauling:

"*Many investigators contributed to demonstrating that it is lipoprotein(a) that is deposited in plaques, not merely LDL, but Lp(a). If you have more than 20 mg/dl in the blood it begins to deposit plaques and cause atherosclerosis. The question then is: What causes Lp(a) to stick to the wall of the artery and form these plaques? "Well countless biochemists and chemists discovered what in the wall of the artery causes Lp(a) to adhere and form atherosclerotic plaques and ultimately lead to heart disease, strokes, and peripheral arterial disease. The answer is that there is a particular amino acid in a protein in the wall of the artery - lysine - which is one of the twenty amino acids that binds the Lp(a) and causes atherosclerotic plaques to develop. I THINK IT IS A VERY IMPORTANT DISCOVERY*" [Linus Pauling, JON, 1994]

Furthermore, Pauling said, "*knowing that lysyl residues are what causes lipoprotein-(a) to get stuck to the wall of the artery and form atherosclerotic plaques, any physical chemist would say at once that the thing to do is prevent that by putting the amino acid lysine in the blood to a greater extent than it is normally. You need lysine to be alive, it is essential, you have to get about one gram a day to keep in protein balance, but you can take lysine, pure lysine, a perfectly non toxic substance in food [as supplements], which puts extra lysine molecules in the blood. They enter into competition with the lysyl residues on the wall of arteries and accordingly count to prevent Lp(a) from being deposited, or even will work to pull it loose and destroy atherosclerotic plaques.*" [Linus Pauling, JON, 1994]

Currently, there are no drugs available for the Lp(a) problem.

At age 92, Pauling lectured on this important develop-ment. After discussing the first three known cases where high

amounts of vitamin C and lysine were successfully used in the treatment of advanced atherosclerosis, Pauling remarked:

"Well, I don't know that there is a need for a randomized prospective, double blind controlled trial when you get evidence of this sort, the value of large intakes of vitamin C and also of lysine for preventing the deposition of atherosclerotic plaques, and preventing death from cardiovascular disease."

SUMMARY

In response to an interviewer's question whether the new therapy of **vitamin C** and large amounts of **lysine** could really reverse the atherosclerotic process?", Dr. Pauling replied:

"I think so. Yes. Now I've got to the point where I think we can get almost complete control of cardiovascular disease, heart attacks and strokes by the proper use of vitamin C and Lysine. It can prevent cardiovascular disease and even cure it. If you are at risk of heart disease, or if there is a history of heart disease in your family, if your father or other members of the family died of a heart attack or stroke or whatever, or if you have a mild heart attack yourself then you had better be taking vitamin C and Lysine." [Linus Pauling, JON, 1994]

MEDLINE

A simple MEDLINE medical database query from the National Institute of Health's internet page will find more than 1100 mainstream studies that mention Lp(a). While confirming Pauling's and Rath's insight, no independent studies (*other than a single study conducted by Dr. Rath which showed great benefit*) has yet formally investigated the use of vitamin C and lysine to eliminate plaque *in human beings.* In 1998 **The Vitamin C Foundation** submitted a study proposal to the United States National

Institutes of Health that is designed to fairly investigate the *Pauling Therapy*. The study proposal was rejected.

Selected MEDLINE Lp(a) ABSTRACTS 1996, 1997

The following few abstracts are examples of the 1100 that were found using MEDLINE during September 1997. **To obtain all 1100 abstracts on DOS/Windows formatted floppies, send $15 to LIPOPROTEIN(A) STUDIES, P. O. Box 3097, Lisle, IL 60532. Please allow 2-3 weeks for delivery.**

MEDLINE CITATION 18
NLM CIT. ID: 97379632
TITLE: Association of an acute reduction in lipoprotein(a) with coronary artery restenosis after percutaneous transluminal coronary angioplasty.
AUTHOR: Horie H; Kinoshita M; Okamura H; Kito O; Sakamoto T
Fujita T; Takaoka A; Izumi M; Takahashi M

AUTHOR AFFILIATION:
First Department of Internal Medicine, Shiga University of Medical Science, Japan.
PUBLICATION TYPES:
JOURNAL ARTICLE
ABSTRACT:

BACKGROUND: Lipoprotein(a) [Lp(a)], which structurally resembles-tissue-type plasminogen, is reported to be associated with coronary atherosclerosis. We examined whether the acute change in Lp(a) by percutaneous transluminal coronary angioplasty (PTCA) is related to restenosis after PTCA. METHODS AND RESULTS:

We measured serum Lp(a) and other lipid parameters (triglycerides and total, LDL, and HDL cholesterol) before and 1 day after PTCA in 143 procedures and 3 days after and 4 months after PTCA in 62 procedures. Quantitative coronary angiography was performed, and restenosis was defined according to three criteria: (1) clinical recurrence of ischemic symptoms, (2) a final stenosis > 50%, and (3) an absolute decrease in minimal lumen diameter > 1/2 of the acute gain in the dilated segment. Restenosis was recognized in 25.9%, 35.7%, and 38.5% of the cases 4 months after PTCA for each criterion, respectively. Although triglyceride and LDL, HDL, and total cholesterol levels were similar in the restenosis and no-restenosis groups before PTCA, Lp(a) was significantly higher in the restenosis group. We found a significant reduction in Lp(a) in the restenosis but not the no-restenosis group 1 day after PTCA. At 3 days after and 4 months after PTCA, Lp(a) was similar in the two groups. A multivariate-analysis revealed that the absolute change in Lp(a)

(before versus 1 day after PTCA) to be the sole significant predictor of restenosis among the clinical, angiographic, and plasma lipid parameters examined.

CONCLUSIONS: **Lp(a) levels were significantly higher in the restenosis group, and they fell significantly after PTCA in the restenosis group.**

SOURCE: Circulation 1997 Jul 1;96(1):166-73

MEDLINE CITATION 27

NLM CIT. ID: 97293308

TITLE: Lipoprotein (a): a potential biological marker for unruptured intracranial aneurysms.

AUTHOR: Phillips J; Collins P; Farrell M; Bouchier-Hayes D el Baghdady A; Bolger C; Roberts G

AUTHOR AFFILIATION:

Department of Neurosurgery, Beaumont Hospital, Dublin, Eire.

PUBLICATION TYPES:

JOURNAL ARTICLE

ABSTRACT:

OBJECTIVE: The diagnosis and treatment of intracranial aneurysms (IAs) prior to rupture reduces the high morbidity and mortality associated with their occurrence. Elevated serum lipoprotein (a) [Lp(a)] level, an independent risk factor for atherogenesis, has been demonstrated in sporadic IA disease (1). The purpose of this study was to assess the degree of correlation between elevated Lp(a) levels and the occurrence of IAs in asymptomatic first degree relatives of index cases from three families exhibiting a familial tendency towards IA development. METHODS: 25 family members and 41 healthy controls were screened by random serum

Lp(a) sampling. All family members received 4-vessel cerebral angiography. RESULTS: Eleven family members were found on angiography to harbour asymptomatic aneurysms and all were successfully treated by surgery. Of these 11, ten had significantly raised serum Lp(a) levels (> 30 mg%). Fourteen family members had negative angiograms. Eight of this latter group, mean age 43.6 +/- 3.8 years, had serum Lp(a) levels above the normal range. Mean Lp(a) levels were 53.7 +/- 1.2 mg% in subjects with aneurysms compared with 22.1 +/- 1.45 mg% in subjects without demonstrable aneurysms and 10.5 +/- 0.48 mg% in the control population. CONCLUSION: **The prevalence of elevated Lp(a) levels in these families and the high degree of association of raised Lp(a) levels with the presence of IAs in several family members warrants follow up of angiographically negative young subjects. We require a case-control study to establish whether particular polymorphisms at the apoprotein (a) gene level are associated with the occurrence of IAs in these families.**

SOURCE: Neurosurgery 1997 May;40(5):1112-5; discussion 1115-7

MEDLINE CITATION 38
NLM CIT. ID: 97276136
TITLE: A prospective study of the role of lipoprotein(a) in the pathogenesis of unstable angina.
AUTHOR: Stubbs P; Noble M; Collinson P; O'Connor B
Moseley D; Seed M
AUTHOR AFFILIATION:
Charing Cross and Westminster Medical School, London, U.K.
PUBLICATION TYPES:
JOURNAL ARTICLE
ABSTRACT:

AIM: Lipoprotein(a) is considered to be a risk factor for atherothrombotic diseases. The aim of the study was to examine the potential role of lipoprotein(a) concentrations in patients admitted with unstable angina with particular reference to their cardiac Troponin T concentrations, which identifies a subgroup at high risk of subsequent cardiac events. METHODS AND RESULTS:

Consecutive patients admitted with chest pain to a single coronary care unit were studied. One hundred and sixty-seven patients had a final diagnosis of unstable angina, of whom 56 were cardiac Troponin T-positive. Admission lipoprotein(a) concentrations were significantly higher in the cardiac Troponin T-positive unstable angina group as compared to the cardiac Troponin T-negative unstable angina group: median and interquartile ranges 22.25 mg.dl-1 (6.25, 32.0) vs 6.0 mg.dl-1 (2.22, 14.8) respectively) P = 0.0004. A highly significant correlation was also found between the level of cardiac Troponin T at diagnosis and the lipoprotein(a) concentration: rs = 0.2798, P = 0.0001. CONCLUSION: **This study provides the first evidence in man of a significant role for lipoprotein(a) in unstable angina.**

The correlation between lipoprotein(a) concentration and cardiac Troponin T concentration suggests that lipoprotein(a) may be significantly involved in the early failure of plaque rupture stabilization.
SOURCE: Eur Heart J 1997 Apr;18(4):603-7

MEDLINE CITATION 48
NLM CIT. ID: 97326406
TITLE: Lipoprotein Lp(a) excess and coronary heart disease [see comments]
AUTHOR: Stein JH; Rosenson RS
COMMENTS:
Comment in: Arch Intern Med 1997 Jun 9;157(11):1161-2
AUTHOR AFFILIATION:
Section of Cardiology, University of Wisconsin Medical School, Madison, USA.
PUBLICATION TYPES:
JOURNAL ARTICLE
REVIEW
REVIEW, TUTORIAL

ABSTRACT:

Lipoprotein Lp(a) excess has been identified as a powerful predictor of premature atherosclerotic vascular disease in several large, prospective studies. Lipoprotein Lp(a) levels modulate the risk of coronary heart disease in patients with hypercholesterolemia, and lipoprotein Lp(a) excess is commonly detected in men and women with premature coronary atherosclerosis. Lipoprotein Lp(a) contributes to atherothrombotic risk by multiple mechanisms that include impaired fibrinolysis, increased cholesterol deposition in the arterial wall, and enhanced oxidation of low density lipoprotein cholesterol. Although low density lipoprotein cholesterol reduction is the primary intervention in patients with lipoprotein Lp(a) excess, specific therapy to lower lipoprotein Lp(a) may be indicated for patients with premature coronary atherosclerosis, a strong family history of premature atherosclerosis, or refractory hypercholesterolemia. In consideration of the high prevalence of lipoprotein Lp(a) excess in patients with premature coronary heart disease and the intricate role of lipoprotein Lp(a) in atherothrombosis, this review provides an evidence-based approach to the screening and treatment of patients with lipoprotein Lp(a) excess.
SOURCE: Arch Intern Med 1997 Jun 9;157(11):1170-6

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Suggested Reading

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2. *Eradicating Heart Disease*, Matthias Rath, Health Now, 1993, ISBN 0-9638768-0-5
3. *Protein Power*, Michael R. Eades and Mary Dan Eades, Bantam Book, 1996, ISBN 0-553-10183-8
4. *Prescription Alternatives*, Earl Mindell and Virginia Hopkins, Keats Publishing, 1998, ISBN 0-87983-790-X
5. *Nutrition Against Disease*, Roger J. Williams, Bantam Book, 1971
6. *The Healing Factor: Vitamin C Against Disease*: Irwin Stone, 1976, Dunlap Books
7. *The Vitamin C Connection,* Emanuel Cheraskin, W. Marshall Ringsdorf, Jr. and Emily L. Sisley, Harper & Row, 1983, ISBN 0-06-038024-1
8. *Cancer and Vitamin C*, Ewan Cameron and Linus Pauling, Camino Books, 1979&1993, ISBN 0-940159-21-X

Final Footnote

Linus Pauling, always a scientist well ahead of his time, may have made the most important health discovery this century. The 1994 Pauling/Rath patent identifies the specific means to disrupt the process of laying down lipoprotein-(a) plaques with chemical agents that make Lp(a) less sticky. These patents are important because they firmly establish the pioneering roles of Linus Pauling and Matthias Rath in this discovery. On the 1993 Unified Theory video Pauling explains how he invented the first "Teflon agent" lysine during one of the first known high-dosage lysine cases. The treatment was successful in a heart patient -- fellow member of the National Academy of Sciences

Thirty years after he turned his scientific talents and attention to nutrition, which incidentally was some 30 years after he began taking large amounts (18,000 mg) of vitamin C every day, Linus Pauling died in August 1994 at the age of 93.

No Government Agency has Sanctioned these Remarks

United States Constitution -- Bill of Rights

1st Amendment

Congress shall make no law respecting an establishment of religion, or prohibiting the free exercise thereof; or abridging the freedom of speech, or of the press; or the right of the people peaceably to assemble, and to petition the government for a redress of grievances.

9th Amendment

The enumeration in the Constitution, of certain rights shall not be construed to deny or disparage others retained by the people

10th Amendment

The powers not delegated to the United States by the Constitution, nor prohibited by it to the States, are reserved to the States respectively, or to the people.

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http//:www.vitaminCfoundation.org

"Hundreds of millions of dollars have been spent by the National Institutes of Health, the American Heart Association, and other agencies in support of studies of cardiovascular disease in relation to LDL and HDL cholesterol, triglycerides, saturated fats, and unsaturated fats. Very little attention has been paid to vitamin C and other vitamins. I think that these agencies have been betting on the wrong horse.

"It is fortunate that vitamin C is not a drug -- it is an orthomolecular substance, normally present in the human body and required for life, and it has extremely low toxicity. You do not need to have a physician's prescription or the approval of the medical establishment to use it in the best way to improve your health and to prevent heart disease. Your knowledge may even be greater and your judgment better than theirs."

--Linus Pauling, *How to Live Longer and Feel Better*, 1986